Periscope.

PATHOLOGY OF NERVOUS SYSTEM.

Histological Changes in the Muscles in Cases of the "Juvenile Form" of Dystrophia Muscularis Progressiva. By W. Erb (*Neurolog. Centrbl.*, July 1, 1886).

In the last number of this Journal we reviewed Erb's monograph on Thomsen's disease, and in that review referred particularly to the histological changes in muscles affected by that disease. In connection with that monograph the above article is of commanding interest. During the past few years Erb has attempted to rearrange and properly classify the large number of diseases included under the term *Progressive Muscular Atrophy*. For those forms of progressive muscular atrophy which are due to disease of the anterior horns of the spinal cord, Erb proposes the name of amyotrophia spinalis progressiva. To this he opposes what he calls cases of *dystrophia muscularis progressiva*, under which head he includes cases of infantile pseudohypertrophy of the muscles, of what Leyden termed hereditary muscular atrophy, and Erb's juvenile muscular atrophy. Duchenne's "Atroph. muscul. progressive de l' enfance" would probably fall under the same head.

It is the "juvenile form" which we are now concerned with; the histological changes of the muscles in this disease have hitherto been but imperfectly known. The condensed history of Erb's case is as follows:

Bohemian Butcher, æt. forty-one, in Erb's clinic from Feb. 19th to March 18, 1886. No hereditary trouble; no syphilis; had conjunctivitis, typhoid, dysentery, carbuncle, hemorrhoids. At the age of thirty-four fell into a quarry to a depth of sixty feet; struck in the small of back by a stone; was unconscious; was in hospital six weeks; no fracture; was able to work after ten weeks. One to one and a half years later present trouble began with weakness in the shoulders, emaciation of upper arm, inability to use arms; is easily fatigued on walking; a feeling of tightness in legs; no other pains in arms or legs; no rigidity; entire nervous system normal. The following changes had taken place in

the muscles: Atrophy and paresis of the pectorals (with exception of small portion of the pars clavicularis), of the trapezii, the rhomboidal, the serrati antic. mag., of both latissimi, of the biceps, brach. antic. and supinator long. of both sides, of both triceps, and of the extensor muscles of the back. There was *hypertrophy* of the deltoids, the flexors of the right forearm, the supra- and infra-spinati, and possibly of the subscapular and of the mm. teretes. Atrophy of left thigh and right gluteal muscles. All other muscles mormal or nearly so. Sensation normal everywhere, cutaneous reflexes normal. Tendon reflexes present but weak. No triceps reflex. Sphincters normal. No fibrillary movements. Mechanical and electrical excitability simply diminished; no R. D.

A small piece of muscle was removed from the hypertrophied right deltoid and from the slightly atrophied right biceps. Deltoid exhibited, on cross-section; all muscular fibres considerably hypertrophied, with the exception of a few that are atrophied. Average diameter 15–170 μ. (normal average 40–60 μ.). The largest ten fibres measured 130–170 μ., the smallest ten fibres 15–40 μ. The fibres are well rounded instead of polygonal, and are farther apart than normally; some fibres exhibit the peculiar vacuolization referred to in the review of "Thomsen's Disease." Connective tissue is slightly increased; the blood-vessels exhibit thickened walls and increased numbers of nuclei. About the same changes are exhibited on longitudinal sections.

The biceps seems to exhibit the disease in its more advanced form; some of the bundles resemble those of the deltoid; other bundles exhibit a smaller number of hypertrophied fibres and a larger number of smaller fibres (very much below the average). The individual fibres are widely separated from one another; vacuolization has not been observed in these. Connective tissue increased and a segmentation of the muscular fibres. Vessels thickened; no fatty infiltration.

The important points to be noticed are that the chief changes are in the muscular fibres themselves and not so much in the connective tissue, and that these changes consist in simple hypertrophy, proliferation of the nuclei, division and vacuolization of the muscular fibres. Later on in the disease simple atrophy is superadded, but there is no fatty or other degeneration of these fibres. The histological changes in the juvenile dystrophy Erb considers very similar to those commonly found in cases of infantile pseudo-hypertrophy. B. S.

De l' Atrophie Musculaire dans les Paralysies Hystériques. Dr. J. Babinsky. *Progrès Médical*, 1886, I., p. 329.

From Charcot's clinic B. reports four cases of muscular atrophy occurring in hysterical paralysis. The absence of trophic symptoms has always been considered a cardinal diagnostic point between paralysis due to hysteria and paralysis due to other causes.

Now, however, in view of the present facts this negative symptom loses part of its value. In two of these cases the paralysis was monoplegic ; in the other two hemiplegic, without facial paralysis. The characteristics of this hysterical muscular atrophy are : 1st. It is more or less extended. 2d. There are no fibrillary twitchings. 3d. The idio-muscular excitability appears to be normal. 4th. Electrical excitability diminished in proportion to the amount of atrophy, but no degeneration reaction. 5th. The atrophy may come on very rapidly. 6th. Its retrogression appears also to be rapid. B. looks upon this atrophy as a simple one, that is not dependent upon any material lesion of the gray matter of the cord or of the peripheral nerves. He also believes it to be distinctly trophic, the nervous system however not presenting any change recognizable by our methods of investigation ; in short, a purely dynamic alteration, analogous to the changes which Charcot believes take place in the spinal cord in the atrophies consecutive to joint lesions. The important part of this communication is not so much in the theory of production as in the fact that an atrophy of a paralyzed muscle may be due to hysteria.

Note Relative à l' Existence de la Névrite Segmentaire Périaxile, à Propos d' un Cas de Paralysie Diphtheritique. A. GOMBAULT. *Progrès Médical*, 1886, p. 472.

A recent article by Pitres and Vaillard in the *Archives de Neurologie* again directs attention to the lesions found in peripheral nerves in diphtheritic paralysis. These investigators found segmentary neuritis, together with lesions indicative of Wallerian degeneration. The segmentary neuritis was not periaxial—that is to say, that in every place in which the myeline was affected the axis-cylinder had disappeared. Gombault now shows that besides this form of neuritis there is also another which is the periaxial one. In the specimens which form the basis of the communication, the axis-cylinder could be traced through the affected part of the nerve and was in direct continuity with the healthy part. It had, howeyer, not completely preserved its normal characteristics. In parts it was swollen and flattened. In the three cases which the author has observed the neuritis was distinctly periaxial.

G. W. JACOBY.

Some Forms of Paralysis Depending upon Peripheral Neuritis. By THOMAS BUZZARD, M. D., *Lancet*, 1885.

Neurologists in Germany and France within the past few years have been devoting considerable attention to the various forms of peripheral neuritis and the paralyses resulting therefrom. An impetus was given to the study of these pathological conditions by an outbreak, within the past few years, of endemic paralysis that occurred among Chinese and Japanese coolies, wherever these laborers were to be found. Scheube was among the first to make

careful autopsies of fatal cases. He almost invariably found an intact brain and cord, while the nerve trunks and branches were in a condition of parenchymatous degeneration. The disease in China and Japan is known as Beri-beri, and has occurred as epidemics in these countries for a great number of years.

Dr. Thomas Buzzard, in the series of Harveian lectures, 1885, treats the subject of paralysis depending upon peripheral neuritis rather from a clinical than from a pathological standpoint (*Lancet*, Nov. 28, Dec. 12 and 19, 1885). A typical example of localized peripheral neuritis is first portrayed, followed by a series of clinical views of cases that have occurred under the author's observation. He distinguishes, pathologically, two forms of neuritis: interstitial neuritis, in which the connective tissue of the nerve is the primary seat of inflammatory changes, the essential element being secondarily affected; and parenchymatous neuritis, in which there is destruction of the essential element of the nerve fibres, with but little or, perhaps, no recognizable alteration in the interstitial tissue." The prominent symptoms for a typical case of peripheral neuritis are the following: marked paralysis, loss or diminished electrical reaction, agonizing pain of a lancinating character, a sodden, œdematous appearance of the affected member, the skin being glossy in patches and exhibiting a purple discoloration and occasionally a bulbous eruption. But cases are not always typical, and even localized neuritis may at times resemble a central disease. An instructive history of such a case is narrated. The diagnosis must then rest chiefly on concomitant circumstances. Electrical examination will afford great assistance in most cases, but the author emphasizes the fact that the resistance offered by the skin and subjacent tissues to the electrical current may sometimes vary in the two sides of the body. The strong relationship between gout and peripheral neuritis must be borne in mind in diagnosticating an obscure case of paralysis. The difficulties encountered in diagnosticating multiple neuritis are extremely great and at times almost impossible. As a rule, however, in multiple neuritis the sphincters of the bladder and rectum remain intact. That form of progressive multiple neuritis due to alcoholic intoxication possesses some special clinical features. The patient usually exhibits some mental disturbance, the memory is especially weakened, and there is a tendency to incoherent talk. Diphtheritic paralysis is held by the author to be dependent upon peripheral neuritis.

It is difficult to do justice to the many valuable and frequent observations that the lectures embody in a short abstract, and a careful perusal of the original is highly commended.

H. N. VINEBERG.

Absence of Patellar Reflex as the only Symptom of Locomotor Ataxia. Société de Biologie. Meeting of April 10, 1886.

DÉJÉRINE reports the case of a tuberculous patient who during life presented absence of the patellar tendon reflex. He had never presented any other symptoms of locomotor ataxia, no lightning pains, no ocular symptoms, no incöordination, etc. At the autopsy, the spinal cord, which was examined with the greatest care, as also the peripheral nerves, were found to be absolutely normal. This case proves that the patellar reflex may be absent without any other symptom, or any characteristic lesion, of locomotor ataxia being present.

Ablation of the Motor Centres. Meeting of April 17, 1886. DUPIN, presents a dog in whom he has removed the motor centers. This dog is able to execute all movements. Laborde, remarks that the dog presents a certain amount of ataxia of gait.

Radial Paralysis due to Compression.—DÉJÉRINE communicates the results of researches made by himself and Vulpain upon six patients affected with radial paralysis due to compression. The cause of this paralysis is always the same. All the muscles are affected, except the triceps, which generally escapes. The duration is always long, at least five to six months. The faradic excitability of the radial nerve is normal. Excitation above the point of compression does not elicit any contraction of the muscles. The nutrition of these muscles is not changed ; they are not at all atrophied, except, perhaps, the supinator longus, which is slightly diminished in volume. Besides this, the muscle presents the signs of degeneration reaction. The main characteristic of this paralysis, then, is a nerve which does not allow the will to pass, but which preserves its faradic excitability.

Vulpian and Déjérine have endeavored to reproduce this paralysis upon animals, but they have not been able to succeed. The question as to the exact nature of the lesion is still an open one. It is certain that the axis-cylinder cannot be affected. Brown-Séquard believes that in these cases it is a peripheral excitation which produces an inhibition upon the spinal cord. The compression, which is insufficient to produce a degeneration of the nerve, is, however, sufficient to produce an effect upon the nervous centres. It is probable that in such patients, secondary alteration of the spinal cord will be found.

Physiological Action of Hypnone.—QUINQUAND presents a communication relative to the action of hypnone upon the blood. As a result of injecting 2 to 3 cubic centimétres of hypnone into the circulation of a dog, an increase of carbonic acid in the arterial blood was found ; also an increased consumption of oxygen. Therefore if a sufficient quantity be used, all the phenomena of asphyxia are produced. All the animals experimented upon died. In practice, therefore. hypnone must be used with the greatest caution.

Alteration of Peripheral Nerves in Chronic Articular Rheumatism. Meeting of June 12, 1886.

PITRES and VALLARD communicate the results of their studies

upon this subject. Chronic articular rheumatism, they say, by its clinical course and symptomatic manifestations, recalls certain forms of trophic affections. It is generally acceded that the nerves are not affected in this disease, but P. and V. now show that this is not always the case. In two of their cases deep and diffuse changes were encountered. These two patients, during life presented all the pronounced symptoms of arthritis deformans, with severe osseous changes and also trophic affections of the skin and nails. The nerves were found to be the seat of parenchymatous neuritis. Whether the peripheral nerves are always thus affected, or whether the neuritis is simply due to a special localization of the rheumatism, are questions which the authors do not attempt to solve. G. W. J.

MENTAL PATHOLOGY.

Paretic Dementia and Cerebral Lues.—Dr. HUGO ENGEL (*Medical Bulletin*, July, 1886) in defence of his position. J. McC——, after having suffered from headache, worse at night, for a considerable time, about a year or so ago, upon one night appearing before the public, had suddenly shown symptoms of aphasia, and of a loss of memory in general. From that time his mental faculties began to decline. After these symptoms of mental decay had been in existence for some time, paresis attacked various groups of muscles, and more decided psychical disturbances made their appearance, but he never exhibited any ideas of magnificence or grandeur. A person standing by the bed of the patient, while the latter was dozing, would have never imagined that he had a very sick man before him. The face looked well nourished, and there was nowhere to be seen that emaciation usually observed in far-advanced cases of paretic dementia. Instead of it, and while his complexion was almost florid, paled somewhat by long confinement, there was noticeable, especially between his brows, that peculiar dirty-yellowish discoloration which he had never found absent in cases of chronic syphilis. All the facts pointed to this malady: the history, its mode of commencement, its progress with the absence of hallucinations of magnificence, the apoplectic seizure, the well-preserved general nutrition, the characteristic discoloration over the forehead. It is unnecessary to say, basing the opinion only on evidence here given by Dr. Engel himself, that he has never seen a case of paretic dementia, and furthermore that he is totally unacquainted with the literature, otherwise he would know that, as a rule, while true paretic dements are well nourished, luetic cases are as frequently of an emaciated type. Furthermore, apoplectic attacks are a characteristic phenomenon. Delusions of grandeur are frequently absent in pure paretic dementia on the one hand, while in syphilitic cases they are as frequently present. The results of treatment neither prove nor disprove any thing, as chronic cerebral syphilis in which secondary degenerative changes